AMENDMENTS TO THE SPECIFICATION

Please replace the paragraph bridging pages 4-5 with the following:

One example of a (murine) monoclonal antibody recognizing EpCAM is Edrecolomab (Panorex™) (Koprowski, Somatic Cell Genet. 1979, 5, 957-971 and Herlyn, Cancer Res., 1980, 40, 717-721; incorporated by reference in its entirety). However, the first administration of Panorex™ during adjuvant immunotherapy of colon cancer led to the development and exacerbation of Wegener's granulomatosis suggesting that Panorex™ should be applied cautiously in a patient with autoimmune disease (Franz, Onkologie 2000, 23, 472-474; incorporated by reference in its entirety). The limitations of Panorex™ are the rapid formation of human anti-mouse antibodies (HAMA), the limited ability to interact by its murine IgG2a Fcγ receptor with human immune effector mechanisms and the short half-life in circulation (Frodin, Cancer Res., 1990, 50, 4866-4871; incorporated by reference in its entirety). Furthermore, the murine antibody caused immediate-type allergic reactions and anaphylaxis upon repeated injection in patients (Riethmüller, Lancet 1994, 343, 1177-1183, Riethmüller, J Clin Oncol., 1998, 16, 1788-1794 and Mellstedt, Annals New York Academy of Sciences 2000, 910, 254-261; each incorporated by reference in their entirety).

At page 6, please replace the paragraph at lines 8-26 with the following:

Several advantageous effects are realizable by using an anti-EpCAM immunoglobulin with a serum half-life of at least 15 days. Most importantly, this relatively long serum half-life implies that the anti-EpCAM immunoglobulin administered as part of the inventive method will not be cleared from the blood as rapidly as another immunoglobulin with a shorter half-life, say that of [[IMG-1]]ING-1 as discussed above. Assuming, then, that an anti-EpCAM immunoglobulin fulfilling the requirements of the immunoglobulin to be used in the method of the invention and an anti-EpCAM immunoglobulin not fulfilling these requirements are both administered to a human simultaneously and in identical absolute amounts, more of the former

immunoglobulin will persist in the serum after a given time than the latter immunoglobulin. In a converse sense, the enhanced persistence in the serum allows less of the anti-EpCAM immunoglobulin used in the inventive method to be administered at one time than would be possible for another anti-EpCAM of shorter serum half life while still maintaining a certain predetermined serum trough level, *i.e.*, while ensuring that the total serum concentration of therapeutic agent never drops below the minimum level determined to be necessary for continued efficacy between two consecutive administrations. This has the advantageous effect that less of the anti-EpCAM immunoglobulin of the method of the invention need be applied in any given dose, thereby eliminating the possibility of or at least mitigating any adverse and/or toxic side effects.

At page 7, please replace the paragraph at lines 8-18 with the following:

While not being bound by theory, the inventors believe that an anti-EpCAM immunoglobulin as used in this aspect of the invention elicits a therapeutic effect based on at least one of two different mechanisms in vivo. One mechanism is known as antibody-dependent cellular cytotoxicity ("ADCC"). In ADCC, a cell ("target cell") which is coated with immunoglobulin is killed by a cell ("effector cell") with Fc receptors which recognize the Fc portion of the immunoglobulin coating the target cell. In most cases, the effector cells participating in ADCC are natural killer ("NK") cells which bear on their surface either the Fc receptor **[[**Fc-□-RIII**]]**Fc-γ-RIII and/or the molecule CD16. In this way, only cells coated with immunoglobulin are killed, so the specificity of cell killing correlates directly with the binding specificity – here, EpCAM – of the immunoglobulin coating such cells.